```
Connecting via Winsock to STN



Welcome to STN International!  Enter x:X

LOGINID:SSPTASXS1656

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2


* * * * * * * * * *  Welcome to STN International  * * * * * * * * * *

 NEWS  1          Web Page for STN Seminar Schedule - N. America
 NEWS  2  AUG 15  CAOLD to be discontinued on December 31, 2008
 NEWS  3  OCT 07  EPFULL enhanced with full implementation of EPC2000
 NEWS  4  OCT 07  Multiple databases enhanced for more flexible patent
                  number searching
 NEWS  5  OCT 22  Current-awareness alert (SDI) setup and editing
                  enhanced
 NEWS  6  OCT 22  WPIDS, WPINDEX, and WPIX enhanced with Canadian PCT
                  Applications
 NEWS  7  OCT 24  CHEMLIST enhanced with intermediate list of
                  pre-registered REACH substances
 NEWS  8  NOV 21  CAS patent coverage to include exemplified prophetic
                  substances identified in English-, French-, German-,
                  and Japanese-language basic patents from 2004-present
 NEWS  9  NOV 26  MARPAT enhanced with FSORT command
 NEWS 10  NOV 26  MEDLINE year-end processing temporarily halts
                  availability of new fully-indexed citations
 NEWS 11  NOV 26  CHEMSAFE now available on STN Easy
 NEWS 12  NOV 26  Two new SET commands increase convenience of STN
                  searching
 NEWS 13  DEC 01  ChemPort single article sales feature unavailable
 NEWS 14  DEC 12  GBFULL now offers single source for full-text
                  coverage of complete UK patent families
 NEWS 15  DEC 17  Fifty-one pharmaceutical ingredients added to PS

 NEWS EXPRESS JUNE 27 08 CURRENT WINDOWS VERSION IS V8.3,
              AND CURRENT DISCOVER FILE IS DATED 23 JUNE 2008.

 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS LOGIN    Welcome Banner and News Items
 NEWS IPC8     For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that
specific topic.

  All use of STN is subject to the provisions of the STN Customer
  agreement.  Please note that this agreement limits use to scientific
  research.  Use for software development or design or implementation
  of commercial gateways or other similar uses is prohibited and may
  result in loss of user privileges and other penalties.

* * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 17:23:26 ON 22 DEC 2008
```

```
=> File Medline EMBASE Biosis Caplus
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.21       0.21

FILE 'MEDLINE' ENTERED AT 17:23:40 ON 22 DEC 2008

FILE 'EMBASE' ENTERED AT 17:23:40 ON 22 DEC 2008
Copyright (c) 2008 Elsevier B.V. All rights reserved.

FILE 'BIOSIS' ENTERED AT 17:23:40 ON 22 DEC 2008
Copyright (c) 2008 The Thomson Corporation

FILE 'CAPLUS' ENTERED AT 17:23:40 ON 22 DEC 2008
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2008 AMERICAN CHEMICAL SOCIETY (ACS)

=> s (casein kinase 1 gamma) or (casein kinase I gamma) or (CSNK1G) or (CSNK1gamma)
or (CSNK1 gamma) or (casein kinase 1, gamma) or (casein kinase I, gamma) or (CSNK1,
gamma)
L1          82 (CASEIN KINASE 1 GAMMA) OR (CASEIN KINASE I GAMMA) OR (CSNK1G)
               OR (CSNK1GAMMA) OR (CSNK1 GAMMA) OR (CASEIN KINASE 1, GAMMA) OR
               (CASEIN KINASE I, GAMMA) OR (CSNK1, GAMMA)

=> S p21 or CIP1 or CDKN1A or (Cyclin-dependent kinase inhibitor 1A)
L2       128374 P21 OR CIP1 OR CDKN1A OR (CYCLIN-DEPENDENT KINASE INHIBITOR 1A)

=> s l1 (P) l2
L3           4 L1 (P) L2

=> duplicate
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
ENTER L# LIST OR (END):l3
DUPLICATE PREFERENCE IS 'EMBASE, BIOSIS, CAPLUS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L3
L4             2 DUPLICATE REMOVE L3 (2 DUPLICATES REMOVED)

=> d l4 1-2 bib ab

L4   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2008 ACS on STN
AN   2004:143261  CAPLUS
DN   140:176313
TI   casein kinase I gamma-1 isoforms
     (CSNK1G1s) as modifiers of the p21 pathway and uses thereof in
     diagnosis, therapy and drug screening
IN   Francis-Lang, Helen; Friedman, Lori; Kidd, Thomas; Roche, Siobhan; Zhang,
     Haiguang
PA   Exelixis, Inc., USA
SO   PCT Int. Appl., 69 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 8
     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
     ----------------    ----   --------    --------------------   --------
PI   WO 2004015071       A2     20040219    WO 2003-US24551        20030806
     WO 2004015071       A3     20040812
          W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
              CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
```

GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

CA	2494236	A1	20040219	CA 2003-2494236	20030806
AU	2003263995	A1	20040225	AU 2003-263995	20030806
EP	1534852	A2	20050601	EP 2003-784937	20030806

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

JP	2005534334	T	20051117	JP 2004-527773	20030806
US	20050251870	A1	20051110	US 2005-523588	20050204
PRAI US	2002-401739P	P	20020807		
	WO 2003-US24551	W	20030806		

AB The invention has designed a dominant loss of function screen to identify genes that interact with the cyclin dependent kinase inhibitor p21 in Drosophila. Casein kinase I gamma-1 isoform 3 (CSNK1G1) gene was identified as a modifier of the p21 pathway. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, casein kinase I gamma-1 isoform (CSNK1G1) genes are attractive drag targets for the treatment of pathologies associated with a defective p21 signaling pathway, such as cancer. The invention also provides methods for utilizing these p21 modifier genes and polypeptides to identify candidate therapeutic agents that can be used in the treatment of disorders associated with defective p21 function.

L4 ANSWER 2 OF 2 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN DUPLICATE 1

AN 1999268046 EMBASE

TI Angiotensin II stimulates serine phosphorylation of the adaptor protein Nck: Physical association with the serine/threonine kinases Pak1 and casein kinase I.

AU Voisin, Laure; Meloche, Sylvain (correspondence)

CS Centre de Recherche, Ctr. Hosp. de l'Univ. de Montreal, University of Montreal, 3850 St. Urbain, Montreal, Que. H2W 1T8, Canada. meloches@ere.umontreal.ca

AU Larose, Louise

CS Department of Experimental Medicine, McGill University, Montreal, Que. H3A 2B2, Canada.

AU Meloche, Sylvain (correspondence)

CS Centre de Recherche, Centre hospitalier Univ. de Montreal, Campus Hotel-Dieu, 3850 St. Urbain, Montreal, Que. H2W 1T8, Canada. meloches@ere.umontreal.ca

SO Biochemical Journal, (1 Jul 1999) Vol. 341, No. 1, pp. 217-223.
Refs: 44
ISSN: 0264-6021 CODEN: BIJOAK

CY United Kingdom

DT Journal; Article

FS 029 Clinical and Experimental Biochemistry

LA English

SL English

ED Entered STN: 12 Aug 1999
Last Updated on STN: 12 Aug 1999

AB Nck is a small adaptor protein consisting exclusively of three SH3 domains and one SH2 domain. Nck is thought to have an important role in cell signalling by coupling receptor tyrosine kinases, via its SH2 domain, to downstream SH3-binding effectors. We report here that angiotensin II,

working through the AT(1) receptor subtype, stimulates the phosphorylation of Nck in rat aortic smooth muscle cells. Phosphopeptide mapping analysis revealed that Nck is phosphorylated on four peptides containing exclusively phosphoserine in quiescent cells. Treatment with angiotensin II resulted in increased phosphorylation of these four peptides, without the appearance of new phosphopeptides. We show that Nck, via its SH3 domains, specifically binds three major phosphoproteins of 95, 82 and 66 kDa both in vitro and in intact cells. Notably, the phosphorylation of these Nck-binding proteins was found to increase in parallel with that of Nck on stimulation by angiotensin II. One candidate for the 66 kDa phosphoprotein is the serine/threonine kinase p21-activated kinase 1 (Pak1), which was found to form a stable complex with Nck in aortic smooth muscle cells. We have also identified the γ2 isoform of casein kinase I as another protein kinase that associates with Nck in these cells. These findings indicate that Nck is a target of G-protein-coupled receptors and suggest a role for Pak1 and casein kinase I-.gamma.2 in downstream signalling or regulation of the AT(1) receptor.

```
=> s proliferation or (cell division) or (mitosis) or apoptosis or (cell cycle) or
(meiosis) or cancer or oncogenesis
L5     4496759 PROLIFERATION OR (CELL DIVISION) OR (MITOSIS) OR APOPTOSIS OR
               (CELL CYCLE) OR (MEIOSIS) OR CANCER OR ONCOGENESIS

=> s l1 (P) l5
L6          23 L1 (P) L5

=> duplicate
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
ENTER L# LIST OR (END):l6
DUPLICATE PREFERENCE IS 'MEDLINE, EMBASE, BIOSIS, CAPLUS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L6
L7           10 DUPLICATE REMOVE L6 (13 DUPLICATES REMOVED)

=> d l7 1-10 bib ab

L7   ANSWER 1 OF 10     MEDLINE on STN                  DUPLICATE 1
AN   2008512208     MEDLINE
DN   PubMed ID: 18694560
TI   A casein kinase 1 and PAR proteins regulate asymmetry of a PIP(2)
     synthesis enzyme for asymmetric spindle positioning.
AU   Panbianco Costanza; Weinkove David; Zanin Esther; Jones David; Divecha
     Nullin; Gotta Monica; Ahringer Julie
CS   The Gurdon Institute and Department of Genetics, University of Cambridge,
     Tennis Court Road, Cambridge CB21QN, UK.
NC   054523 (United Kingdom Wellcome Trust)
SO   Developmental cell, (2008 Aug) Vol. 15, No. 2, pp. 198-208.
     Journal code: 101120028. ISSN: 1534-5807.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
     (RESEARCH SUPPORT, N.I.H., EXTRAMURAL)
     (RESEARCH SUPPORT, NON-U.S. GOV'T)
LA   English
FS   Priority Journals
EM   200809
ED   Entered STN: 13 Aug 2008
     Last Updated on STN: 7 Sep 2008
     Entered Medline: 5 Sep 2008
AB   Spindle positioning is an essential feature of asymmetric cell
```

division. The conserved PAR proteins together with heterotrimeric G proteins control spindle positioning in animal cells, but how these are linked is not known. In C. elegans, PAR protein activity leads to asymmetric spindle placement through cortical asymmetry of Galpha regulators GPR-1/2. Here, we establish that the casein kinase 1 gamma CSNK-1 and a PIP(2) synthesis enzyme (PPK-1) transduce PAR polarity to asymmetric Galpha regulation. PPK-1 is posteriorly enriched in the one-celled embryo through PAR and CSNK-1 activities. Loss of CSNK-1 causes uniformly high PPK-1 levels, high symmetric cortical levels of GPR-1/2 and LIN-5, and increased spindle pulling forces. In contrast, knockdown of ppk-1 leads to low GPR-1/2 levels and decreased spindle forces. Furthermore, loss of CSNK-1 leads to increased levels of PIP(2). We propose that asymmetric generation of PIP(2) by PPK-1 directs the posterior enrichment of GPR-1/2 and LIN-5, leading to posterior spindle displacement.

L7 ANSWER 2 OF 10 MEDLINE on STN DUPLICATE 2
AN 2006124588 MEDLINE
DN PubMed ID: 16247451
TI RNAi-based screening of the human kinome identifies Akt-cooperating kinases: a new approach to designing efficacious multitargeted kinase inhibitors.
AU Morgan-Lappe S; Woods K W; Li Q; Anderson M G; Schurdak M E; Luo Y; Giranda V L; Fesik S W; Leverson J D
CS Abbott Laboratories, Cancer Research, Abbott Park, IL 60064, USA.
SO Oncogene, (2006 Mar 2) Vol. 25, No. 9, pp. 1340-8.
Journal code: 8711562. ISSN: 0950-9232.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200604
ED Entered STN: 3 Mar 2006
Last Updated on STN: 19 Apr 2006
Entered Medline: 18 Apr 2006
AB Tumors comprise genetically heterogeneous cell populations, whose growth and survival depend on multiple signaling pathways. This has spurred the development of multitargeted therapies, including small molecules that can inhibit multiple kinases. A major challenge in designing such molecules is to determine which kinases to inhibit in each cancer to maximize efficacy and therapeutic index. We describe an approach to this problem implementing RNA interference technology. In order to identify Akt-cooperating kinases, we screened a library of kinase-directed small interfering RNAs (siRNAs) for enhanced cancer cell killing in the presence of Akt inhibitor A-443654. siRNAs targeting casein kinase I gamma 3 (CSNK1G3) or the inositol polyphosphate multikinase (IPMK) significantly enhanced A-443654-mediated cell killing, and caused decreases in Akt Ser-473 and ribosomal protein S6 phosphorylation. Small molecules targeting CSNK1G3 and/or IPMK in addition to Akt may thus exhibit increased efficacy and have the potential for improved therapeutic index.

L7 ANSWER 3 OF 10 MEDLINE on STN DUPLICATE 3
AN 2005658616 MEDLINE
DN PubMed ID: 16341016
TI Casein kinase 1 gamma couples Wnt receptor activation to cytoplasmic signal transduction.
AU Davidson Gary; Wu Wei; Shen Jinlong; Bilic Josipa; Fenger Ursula; Stannek Peter; Glinka Andrei; Niehrs Christof
CS Division of Molecular Embryology, Deutsches Krebsforschungszentrum, Im Neuenheimer Feld 280, D-69120 Heidelberg, Germany..

g.davidson@dkfz-heidelberg.de
SO Nature, (2005 Dec 8) Vol. 438, No. 7069, pp. 867-72.
Journal code: 0410462. E-ISSN: 1476-4687.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
OS GENBANK-DQ185136
EM 200512
ED Entered STN: 18 Dec 2005
Last Updated on STN: 30 Dec 2005
Entered Medline: 29 Dec 2005
AB Signalling by Wnt proteins (Wingless in Drosophila) has diverse roles during embryonic development and in adults, and is implicated in human diseases, including cancer. LDL-receptor-related proteins 5 and 6 (LRP5 and LRP6; Arrow in Drosophila) are key receptors required for transmission of Wnt/beta-catenin signalling in metazoa. Although the role of these receptors in Wnt signalling is well established, their coupling with the cytoplasmic signalling apparatus remains poorly defined. Using a protein modification screen for regulators of LRP6, we describe the identification of Xenopus Casein kinase 1 gamma (CK1gamma), a membrane-bound member of the CK1 family. Gain-of-function and loss-of-function experiments show that CK1gamma is both necessary and sufficient to transduce LRP6 signalling in vertebrates and Drosophila cells. In Xenopus embryos, CK1gamma is required during anterio-posterior patterning to promote posteriorizing Wnt/beta-catenin signalling. CK1gamma is associated with LRP6, which has multiple, modular CK1 phosphorylation sites. Wnt treatment induces the rapid CK1gamma-mediated phosphorylation of these sites within LRP6, which, in turn, promotes the recruitment of the scaffold protein Axin. Our results reveal an evolutionarily conserved mechanism that couples Wnt receptor activation to the cytoplasmic signal transduction apparatus.

L7 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2004:143261 CAPLUS
DN 140:176313
TI casein kinase I gamma-1 isoforms (CSNK1G1s) as modifiers of the p21 pathway and uses thereof in diagnosis, therapy and drug screening
IN Francis-Lang, Helen; Friedman, Lori; Kidd, Thomas; Roche, Siobhan; Zhang, Haiguang
PA Exelixis, Inc., USA
SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 8

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004015071	A2	20040219	WO 2003-US24551	20030806
	WO 2004015071	A3	20040812		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	CA 2494236	A1	20040219	CA 2003-2494236	20030806
	AU 2003263995	A1	20040225	AU 2003-263995	20030806
	EP 1534852	A2	20050601	EP 2003-784937	20030806
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	JP 2005534334	T	20051117	JP 2004-527773	20030806
	US 20050251870	A1	20051110	US 2005-523588	20050204
PRAI	US 2002-401739P	P	20020807		
	WO 2003-US24551	W	20030806		

AB The invention has designed a dominant loss of function screen to identify genes that interact with the cyclin dependent kinase inhibitor p21 in Drosophila. Casein kinase I gamma -1 isoform 3 (CSNK1G1) gene was identified as a modifier of the p21 pathway. Accordingly, vertebrate orthologs of these modifiers, and preferably the human orthologs, casein kinase I gamma-1 isoform (CSNK1G1) genes are attractive drag targets for the treatment of pathologies associated with a defective p21 signaling pathway, such as cancer. The invention also provides methods for utilizing these p21 modifier genes and polypeptides to identify candidate therapeutic agents that can be used in the treatment of disorders associated with defective p21 function.

L7 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2004:219931 CAPLUS
DN 140:248186
TI Use of patterns of gene expression to identify tissue types and in disease diagnosis and prognosis
IN Glinskii, Guennadi V.
PA Sidney Kimmel Cancer Center, USA
SO U.S. Pat. Appl. Publ., 209 pp., which which which which
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 20040053317	A1	20040318	US 2003-660434	20030910
	CA 2498418	A1	20040325	CA 2003-2498418	20030910
	WO 2004025258	A2	20040325	WO 2003-US28707	20030910
	WO 2004025258	A3	20050519		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2003274970	A1	20040430	AU 2003-274970	20030910
	EP 1552293	A2	20050713	EP 2003-759240	20030910
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	US 20050142573	A1	20050630	US 2004-861003	20040603
PRAI	US 2002-410018P	P	20020910		
	US 2002-411155P	P	20020916		
	US 2002-429168P	P	20021125		
	US 2003-444348P	P	20030131		
	US 2003-460826P	P	20030403		
	US 2003-660434	A1	20030910		

WO 2003-US28707 W 20030910
AB Methods of using quant. anal. of array hybridizations to identify normal and diseased tissue in the diagnosis and prognosis of disease are described. The methods segregate individual samples into distinct classes using quant. measurements of expression values for selected sets of genes in individual samples compared to a reference standard Samples displaying pos. and neg. correlations of the gene expression values with the reference standard samples exhibit distinct behaviors and pathohistol. features. Also disclosed are methods for identifying sets of genes whose expression patterns are correlated with a phenotype. Such sets are useful for characterizing cellular differentiation pathways and states and for identifying potential drug discovery targets. Panels for diagnosis and determination of risk of invasive and metastatic forms of lung, prostate and breast cancer are identified. Similarly, panels indicating recurrence of the cancers and poor prognostic outcomes are identified.

L7 ANSWER 6 OF 10 MEDLINE on STN DUPLICATE 4
AN 2004279258 MEDLINE
DN PubMed ID: 15077195
TI Metastatic tumor antigen 1 short form (MTA1s) associates with casein kinase I-gamma2, an estrogen-responsive kinase.
AU Mishra Sandip K; Yang Zhibo; Mazumdar Abhijit; Talukder Amjad H; Larose Louise; Kumar Rakesh
CS Department of Molecular and Cellular Oncology, The University of Texas MD Anderson Cancer Center, Houston, TX 77030, USA.
NC CA098823 (United States NCI)
CA90970 (United States NCI)
SO Oncogene, (2004 May 27) Vol. 23, No. 25, pp. 4422-9.
Journal code: 8711562. ISSN: 0950-9232.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LA English
FS Priority Journals
EM 200407
ED Entered STN: 6 Jun 2004
Last Updated on STN: 2 Jul 2004
Entered Medline: 1 Jul 2004
AB Recent studies have shown that metastasis-associated protein-1 short form (MTA1s) - metastatic tumor antigen 1 short form sequesters estrogen receptor-alpha (ER-alpha) in the cytoplasm of breast cancer cells. Using a yeast two-hybrid screening to clone MTA1s-interacting proteins, we identified casein kinase I-gamma 2 (CKI-gamma2, a ubiquitously expressed cytoplasmic kinase) as an MTA1s-binding protein. We show that MTA1s interacts with CKI-gamma2 both in vitro and in vivo and colocalizes in the cytoplasm. In addition, we found that CKI-gamma2 can phosphorylate MTA1s, but not ER, in an antiestrogen-dependent manner and that estrogen stimulates CKI-gamma2 activity that could be effectively blocked by a specific inhibitor of CKI. CKI-gamma2 could further potentiate the ER corepressive function of MTA1s. Kinase dead CK1-gamma2 could not repress estrogen-induced ER transactivation functions. Results from mutagenesis studies suggest that substitution of the serine residue at 321 to alanine, which is a possible CKI-gamma2 phopshorylation site in MTA1s, results in a significant reduction in the ability of MTA1s to repress ER transactivation. These findings identified MTA1s as a target of CKI-gamma2, and provided new evidence to suggest that CKI-gamma2 phosphorylates and modulates the functions of MTA1s, and that these extranuclear effects of estrogen might

have important implications in regulating the functions of MTA1s in human mammary epithelial and cancer cells.

L7 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2003:633500 CAPLUS
DN 139:192515
TI Proteins and genes involved in the regulation of energy homeostasis and triglyceride metabolism and their use in the diagnosis and treatment of metabolic disorders
IN Eulenberg, Karsten; Broenner, Guenter; Steuernagel, Arndt; Meise, Martin; Haeder, Thomas
PA Develogen Aktiengesellschaft Fuer Entwicklungsbiologische Forschung, Germany
SO PCT Int. Appl., 157 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003066086	A2	20030814	WO 2003-EP1094	20030204
	WO 2003066086	A3	20040122		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2003226973	A1	20030902	AU 2003-226973	20030204
PRAI	EP 2002-2548	A	20020204		
	EP 2002-2707	A	20020206		
	EP 2002-2891	A	20020208		
	EP 2002-3748	A	20020219		
	EP 2002-4667	A	20020228		
	EP 2002-22101	A	20021002		
	WO 2003-EP1094	W	20030204		

AB The present invention discloses casein kinase 1.gamma. (CSNK1G), GABAA receptor-associated protein (GABARAP), proliferation-associated 2G4 protein (PA2G4, also referred to as methionyl aminopeptidase homologous protein), molybdenum cofactor synthesis-step 1 protein (MOCS1), cell division cycle 10 protein homolog (CDC10, also referred to as septin and septin 7), pyruvate kinase (PK), calreticulin (CALR), and polynucleotides which identify and encode these proteins. These proteins are shown to be involved in the regulation of energy homeostasis, body weight regulation, and triglyceride metabolism by measuring triglyceride levels in a genetic adipose pathway screen, expression profiling, and synthesis of lipids during adipogenesis. The genetic screen demonstrates that mutations of these genes cause obesity, reflected by a significant increase of triglyceride content, the major energy storage substance. Thus, the invention relates to the use of these sequences in the diagnosis, study, prevention, and treatment of metabolic diseases and disorders.

L7 ANSWER 8 OF 10 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN DUPLICATE 5
AN 2003237680 EMBASE

TI Downregulation of Cap43 gene by von Hippel-Lindau tumor suppressor protein in human renal cancer cells.
AU Masuda, Katsuaki; Ono, Mayumi (correspondence); Okamoto, Masahiro; Morikawa, Wataru; Otsubo, Michihiro; Kuwano, Michihiko
CS Department of Medical Biochemistry, Graduate School of Medical Sciences, Kyushu University, 3-1-1 Maidashi, Higashiku, Fukuoka, 812-8582, Japan. mayumi@biochem1.med.kyushu-u.ac.jp
AU Migita, Toshiro; Tsuneyosh, Masazumi
CS Department of Anatomical Pathology, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan.
AU Okuda, Heiwa; Shuin, Taro
CS Department of Urology, Kochi Medical School, Kochi, Japan.
AU Naito, Seiji
CS Department of Urology, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan.
SO International Journal of Cancer, (20 Jul 2003) Vol. 105, No. 6, pp. 803-810.
Refs: 38
ISSN: 0020-7136 CODEN: IJCNAW
CY United States
DT Journal; Article
FS 016 Cancer
022 Human Genetics
028 Urology and Nephrology
005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 3 Jul 2003
Last Updated on STN: 3 Jul 2003
AB We previously identified 9 genes (i.e., thymosin β4, secreted protein acidic and rich in cysteine, Cap43, ceruloplasmin, serum amyloid A, heat shock protein 90, LOT1, osteopontin and casein kinase 1.gamma.) that are more highly expressed in cancerous regions than in noncancerous regions in human renal cancers. In our study, we considered the possibility that the von Hippel-Lindau (VHL) tumor suppressor gene might be able to affect the expression of these 9 genes in renal cancer cells. We first established 2 VHL-positive cell lines, 786/VHL-1 and 786/VHL-2, after the introduction of wild-type VHL into VHL-negative renal cancer 786-0 cells. Of these 9 genes, expression of the Cap43 gene was specifically downregulated by VHL. Expression of Cap43 was also much lower in 4 other VHL-positive renal cancer cell lines than in VHL-negative 786-0 cells. Cap43 promoter assays with several deletion or mutation constructs demonstrated that the Sp1 site in the element from -286 base pairs (bp) to -62 bp was partly responsible for VHL-induced suppression of the Cap43 gene. Immunostaining analysis with human specimens of renal cancers demonstrated that the Cap43 protein was expressed in most cancer cells and macrophages. We also observed a marked and specific increase of Cap43 mRNA levels in response to hypoxia or nickel in all VHL-positive cell lines. Cellular expression of Cap43 mRNA in response to hypoxia or nickel thus is closely associated with VHL gene expression in renal cancer cells. Although the function of the Cap43 protein remains unclear, the expression of Cap43 protein could be a molecular marker closely associated with VHL in renal cancer. .COPYRGT. 2003 Wiley-Liss, Inc.

L7 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2003:807997 CAPLUS
DN 140:126171
TI Genes commonly upregulated by hypoxia in human breast cancer cells MCF-7 and MDA-MB-231

AU Bando, Hiroko; Toi, Masakazu; Kitada, Kunio; Koike, Morio
CS Breast Cancer Research Group, Tokyo Metropolitan Cancer and Infectious Diseases Center, Bunkyo-ku, Tokyo, 113-0087, Japan
SO Biomedicine & Pharmacotherapy (2003), 57(8), 333-340
CODEN: BIPHEX; ISSN: 0753-3322
PB Editions Scientifiques et Medicales Elsevier
DT Journal
LA English
AB Hypoxia is a stress that causes alterations in signal transduction and gene instability. In the cancer microenvironment, hypoxia plays a significant role in forming a tumor phenotype and tumor progression. We aimed to identify the genes upregulated by hypoxia in human breast cancer cell lines, a hormone-dependent MCF-7 and a hormone-independent MDA-MB-231, using microarray anal. These cells were exposed to two oxygen concns. such as 21% and 1% in a time-course. Out of 12,625 genes, 26 genes were identified as commonly upregulated in both MCF-7 and MDA-MB-231 cells. Some of these genes were already reported as hypoxia-related, but some of those were identified newly. These commonly upregulated genes between hormone-dependent and hormone-independent cells would be a clue to study hypoxia-related events and to explore the novel therapeutic targets in human breast cancer.
RE.CNT 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2002:440558 CAPLUS
DN 137:260870
TI CXCR4/CXCL12 expression and signaling in kidney cancer
AU Schrader, A. J.; Lechner, O.; Templin, M.; Dittmar, K. E. J.; Machtens, S.; Mengel, M.; Probst-Kepper, M.; Franzke, A.; Wollensak, T.; Gatzlaff, P.; Atzpodien, J.; Buer, J.; Lauber, J.
CS Department of Cell Biology and Immunology, German Research Centre for Biotechnology (GBF), Brounschweig, D-38124, Germany
SO British Journal of Cancer (2002), 86(8), 1250-1256
CODEN: BJCAAI; ISSN: 0007-0920
PB Nature Publishing Group
DT Journal
LA English
AB CXCL12 (SDF-1), a CXC-chemokine, and its specific receptor, CXCR4, have recently been shown to be involved in tumorigenesis, proliferation and angiogenesis. Therefore, we analyzed CXCL12α/CXCR4 expression and function in four human kidney cancer cell lines (A-498, CAKI-1, CAKI-2, HA-7), 10 freshly harvested human tumor samples and corresponding normal kidney tissue. While none of the analyzed tumor cell lines expressed CXCL12α, A-498 cells were found to express CXCR4. More importantly, real-time RT-PCR anal. of 10 tumor samples and resp. adjacent normal kidney tissue disclosed a distinct and divergent downregulation of CXCL12α and upregulation of CXCR4 in primary tumor tissue. To prove that the CXCR4 protein is functionally active, rhCXCL12α was investigated for its ability to induce changes of intracellular calcium levels in A-498 cells. Moreover, we used cDNA expression arrays to evaluate the biol. influence of CXCL12α. Comparing gene expression profiles in rhCXCL12α stimulated vs. unstimulated A-498 kidney cancer cells revealed specific regulation of 31 out of 1176 genes tested on a selected human cancer array, with a prominent stimulation of genes involved in cell-cycle regulation and apoptosis. The genetic changes reported here should provide new insights into the developmental paths leading to tumor progression and may also aid the design of new approaches to therapeutic intervention.
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT